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Search Results - Record(s) 11 through 13 of 13 returned.

☐ 11. Document ID: MX 2000007292 A1 WO 9938955 A2 AU 9922911 A BR 9907283 A EP 1049769 A2 GB 2353529 A CN 1295612 A KR 2001040430 A JP 2002501742 W AU 749017 B

L3: Entry 11 of 13 File: DWPI Oct 1, 2001

DERWENT-ACC-NO: 1999-469325
DERWENT-WEEK: 200274
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TITLE: New Herpes Simplex Virus mutant useful for gene therapy

INVENTOR: COFFIN, R S; FINNIE, N J ; LATCHMAN, D S

PRIORITY-DATA: 1998GB-0001930 (January 29, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
MX 2000007292 A1	October 1, 2001		000	A61K048/00
WO 9938955 A2	August 5, 1999	E	020	C12N007/00
AU 9922911 A	August 16, 1999		000	C12N007/00
BR 9907283 A	October 24, 2000		000	C12N007/00
EP 1049769 A2	November 8, 2000	E	000	C12N007/00
GB 2353529 A	February 28, 2001		000	C12N007/00
CN 1295612 A	May 16, 2001		000	C12N007/00
KR 2001040430 A	May 15, 2001		000	A61K039/245
JP 2002501742 W	January 22, 2002		032	C12N007/04
AU 749017 B	June 13, 2002		000	C12N007/00

INT-CL (IPC): A61 K 35/76; A61 K 38/00; A61 K 39/245; A61 K 48/00; A61 P 25/00; C12 N 7/00; C12 N 7/04; C12 N 15/09; C12 N 15/86

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 12. Document ID: WO 9830707 A2 AU 749064 B AU 9855669 A BR 9806866 A CN 1250480 A EP 1021553 A2 MX 9906454 A1 KR 2000070037 A JP 2001508294 W

L3: Entry 12 of 13 File: DWPI Jul 16, 1998

DERWENT-ACC-NO: 1998-399151
DERWENT-WEEK: 200252
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TITLE: Vector comprising herpes simplex virus latency-associated transcript P2 region - useful for treating e.g. Parkinson's disease, spinal injury, stroke, etc

INVENTOR: COFFIN, R S; LATCHMAN, D S

PRIORITY-DATA: 1997GB-0000411 (January 10, 1997)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9830707 A2	July 16, 1998	E	026	C12N015/86
AU 749064 B	June 20, 2002		000	C12N015/86
AU 9855669 A	August 3, 1998		000	C12N015/86
BR 9806866 A	April 18, 2000		000	C12N015/86
CN 1250480 A	April 12, 2000		000	C12N015/86
EP 1021553 A2	July 26, 2000	E	000	C12N015/86
MX 9906454 A1	April 1, 2000		000	C12N015/86
KR 2000070037 A	November 25, 2000		000	C12N015/86
JP 2001508294 W	June 26, 2001		035	C12N015/09

INT-CL (IPC): A61 K 35/76; A61 K 39/00; A61 K 39/245; A61 K 48/00; A61 P 9/00; A61 P 25/00; A61 P 25/16; A61 P 27/02; A61 P 31/22; C12 N 7/00; C12 N 7/01; C12 N 15/09; C12 N 15/85; C12 N 15/86; C12 Q 1/68; C12 N 15/09; C12 R 1:92

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
Draw. Desc | Image

☐ 13. Document ID: WO 9804726 A1 US 6248320 B1 AU 9737007 A EP 920523 A1 AU 726645 B JP 2000516809 W NZ 333901 A

L3: Entry 13 of 13 File: DWPI Feb 5, 1998

DERWENT-ACC-NO: 1998-130712
DERWENT-WEEK: 200137

TITLE: Herpes simplex virus lacking functional ICP34.5 and ICP27 genes - useful for, e.g. treating injuries to central nervous system such as Parkinson's disease and for gene therapy in mammals

INVENTOR: BROWN, S M; COFFIN, R S ; LATCHMAN, S D ; MACLEAN, A R ; LATCHMAN, D S

PRIORITY-DATA: 1996GB-0015794 (July 26, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9804726 A1	February 5, 1998	E	030	C12N015/86
US 6248320 B1	June 19, 2001		000	A01N063/00
AU 9737007 A	February 20, 1998		000	C12N015/86
EP 920523 A1	June 9, 1999	E	000	C12N015/86
AU 726645 B	November 16, 2000		000	C12N015/86
JP 2000516809 W	December 19, 2000		033	C12N015/00
NZ 333901 A	May 25, 2001		000	C12N007/01

INT-CL (IPC): A01 N 63/00; A61 K 35/30; A61 K 35/34; A61 K 48/00; A61 P 25/00; A61 P 25/16; A61 P 35/00; C07 K 14/035; C12 N 1/21; C12 N 7/00; C12 N 7/01; C12 N 7/04; C12 N 15/00; C12 N 15/86; C12 N 1/21; C12 R 1:01

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
Draw. Desc | Image

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Terms	Documents
Coffin R S.in.	13

Display Format: CIT Change Format

Previous Page Next Page

WEST

Generate Collection | Print

Search Results - Record(s) 1 through 10 of 13 returned.

☐ 1. Document ID: GB 2376687 A WO 200177358 A2 AU 200146728 A US 20020099006 A1

L3: Entry 1 of 13 File: DWPI Dec 24, 2002

DERWENT-ACC-NO: 2002-017464
DERWENT-WEEK: 200302
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TITLE: Use of attenuated herpes virus that lacks a functional virion host shut-off protein gene and comprises a functional UL43 gene, in the manufacture of a medicament for stimulating immune response

INVENTOR: COFFIN, R S

PRIORITY-DATA: 2000GB-0009079 (April 12, 2000), 1998GB-0016781 (July 31, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
GB 2376687 A	December 24, 2002		000	C12N015/869
WO 200177358 A2	October 18, 2001	E	035	C12N015/869
AU 200146728 A	October 23, 2001		000	C12N015/869
US 20020099006 A1	July 25, 2002		000	A61K048/00

INT-CL (IPC): A01 N 63/00; A61 K 38/16; A61 K 39/12; A61 K 39/245; A61 K 39/255; A61 K 39/265; A61 K 39/27; A61 K 48/00 ; C07 H 21/04; C12 N 15/869; C12 Q 1/70; C12 N 15/86

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

☐ 2. Document ID: US 20020192802 A1 WO 200153507 A1 AU 200126954 A EP 1250446 A1

L3: Entry 2 of 13 File: DWPI Dec 19, 2002

DERWENT-ACC-NO: 2001-442264
DERWENT-WEEK: 200303
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TITLE: New herpes virus strain capable of replicating in permissive cells which comprises a mutation which results in enhanced ICP0 expression compared to the parental virus for treatment of cancer and tumors

INVENTOR: COFFIN, R S

PRIORITY-DATA: 2000GB-0001476 (January 21, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20020192802 A1	December 19, 2002		000	A61K048/00
WO 200153507 A1	July 26, 2001	E	025	C12N015/869
AU 200126954 A	July 31, 2001		000	C12N015/869
EP 1250446 A1	October 23, 2002	E	000	C12N015/869

INT-CL (IPC): A61 K 39/245; A61 K 39/255; A61 K 39/265; A61 K 39/27; A61 K 48/00; C12 N 7/00; C12 N 15/869

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

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☐ 3. Document ID: BR 200107736 A WO 200153506 A2 AU 200126951 A EP 1252323 A2 GB 2375113 A

L3: Entry 3 of 13 File: DWPI Nov 19, 2002

DERWENT-ACC-NO: 2001-442263
DERWENT-WEEK: 200305
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TITLE: Novel viral strain, used to treat or prevent cancer or parasitic infection, is modified, non-laboratory and optionally oncolytic

INVENTOR: COFFIN, R S

PRIORITY-DATA: 2001GB-0000430 (January 6, 2001), 2000GB-0001475 (January 21, 2000), 2000GB-0002854 (February 8, 2000), 2001GB-0000288 (January 5, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
BR 200107736 A	November 19, 2002		000	C12N015/869
WO 200153506 A2	July 26, 2001	E	048	C12N015/869
AU 200126951 A	July 31, 2001		000	C12N015/869
EP 1252323 A2	October 30, 2002	E	000	C12N015/869
GB 2375113 A	November 6, 2002		000	C12N015/869

INT-CL (IPC): A61 K 48/00; C12 N 7/01; C12 N 15/869; C12 Q 1/02; A61 K 49/00; A61 P 35/00; C12 N 7/01; C12 Q 1/02

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

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☐ 4. Document ID: BR 200107737 A WO 200153505 A2 AU 200126947 A GB 2374873 A EP 1252322 A2

L3: Entry 4 of 13 File: DWPI Nov 19, 2002

DERWENT-ACC-NO: 2001-442262
DERWENT-WEEK: 200305
COPYRIGHT 2003 DERWENT INFORMATION LTD
TITLE: A herpes virus comprising a gene encoding an immunomodulatory protein and lacking a functional ICP34.5 or ICP47 encoding gene for the treatment of cancer

INVENTOR: COFFIN, R S

PRIORITY-DATA: 2001GB-0000430 (January 6, 2001), 2000GB-0001475 (January 21, 2000), 2000GB-0002854 (February 8, 2000), 2001GB-0000288 (January 5, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
BR 200107737 A	November 19, 2002		000	C12N015/869
WO 200153505 A2	July 26, 2001	E	020	C12N015/869
AU 200126947 A	July 31, 2001		000	C12N015/869
GB 2374873 A	October 30, 2002		000	C12N015/869
EP 1252322 A2	October 30, 2002	E	000	C12N015/869

INT-CL (IPC): A61 K 48/00; C12 N 7/01; C12 N 15/869

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

☐ 5. Document ID: EP 1246930 A1 WO 200146450 A1 AU 200122088 A

L3: Entry 5 of 13 File: DWPI Oct 9, 2002

DERWENT-ACC-NO: 2001-441680
DERWENT-WEEK: 200267
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TITLE: Use of replication incompetent virus comprising heterologous gene for manufacturing medicament to treat or prevent peripheral nervous system disorder, treating motor neuron disease, peripheral nerve damage in subject

INVENTOR: COFFIN, R S

PRIORITY-DATA: 1999GB-0030419 (December 22, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1246930 A1	October 9, 2002	E	000	C12N015/869
WO 200146450 A1	June 28, 2001	E	036	C12N015/869
AU 200122088 A	July 3, 2001		000	C12N015/869

INT-CL (IPC): A61 K 48/00; C12 N 7/04; C12 N 15/869

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

☐ 6. Document ID: EP 1240344 A1 WO 200146449 A1 AU 200120175 A

L3: Entry 6 of 13 File: DWPI Sep 18, 2002

DERWENT-ACC-NO: 2001-408653
DERWENT-WEEK: 200269
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TITLE: Use of replication incompetent herpes virus having mutation that prevents 2 immediate early genes expression, and heterologous gene linked to promoter active during herpes virus latency, to treat Parkinson's disease

INVENTOR: COFFIN, R S

PRIORITY-DATA: 1999GB-0030418 (December 22, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1240344 A1	September 18, 2002	E	000	C12N015/86
WO 200146449 A1	June 28, 2001	E	042	C12N015/86
AU 200120175 A	July 3, 2001		000	C12N015/86

INT-CL (IPC): A61 K 48/00; A61 P 25/00; A61 P 25/16; A61 P 25/28; C12 N 15/86; G01 N 33/50

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

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☐ 7. Document ID: JP 2002526039 W WO 200008194 A2 AU 9951837 A BR 9912719 A EP 1102858 A2 GB 2361243 A KR 2001072236 A CN 1321199 A

L3: Entry 7 of 13 File: DWPI Aug 20, 2002

DERWENT-ACC-NO: 2000-205732
DERWENT-WEEK: 200258
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TITLE: Propagation of a mutant herpes simplex virus to treat e.g. Parkinson's disease, comprises infecting a cell line with a mutated endogenous HSV viral protein 16 gene capable of undergoing homologous recombination with the endogenous gene

INVENTOR: COFFIN, R S; LATCHMAN, D S

PRIORITY-DATA: 1998GB-0016856 (August 3, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2002526039 W	August 20, 2002		037	C12N015/09
WO 200008194 A2	February 17, 2000	E	027	C12N015/86
AU 9951837 A	February 28, 2000		000	C12N015/86
BR 9912719 A	May 2, 2001		000	C12N015/86
EP 1102858 A2	May 30, 2001	E	000	C12N015/86
GB 2361243 A	October 17, 2001		000	C12N015/86
KR 2001072236 A	July 31, 2001		000	C12N007/00
CN 1321199 A	November 7, 2001		000	C12N015/86

INT-CL (IPC): A61 K 35/76; A61 K 48/00; C12 N 5/10; C12 N 7/00; C12 N 7/04; C12 N 15/09; C12 N 15/86; C12 N 5/10; C12 N 7/04; C12 R 1:91; C12 R 1:93

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

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☐ 8. Document ID: WO 200008191 A2 AU 9951822 A BR 9912653 A EP 1100942 A2 GB 2361921 A KR 2001072162 A US 20020099006 A1

L3: Entry 8 of 13 File: DWPI Feb 17, 2000

DERWENT-ACC-NO: 2000-205729
DERWENT-WEEK: 200255
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TITLE: Viral vectors useful for treating viral infection and cancer, comprises

attenuated herpes simplex virus capable of infecting dendritic cell

INVENTOR: CHAIN, B; COFFIN, R S

PRIORITY-DATA: 1998GB-0016781 (July 31, 1998), 2000GB-0009079 (April 12, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200008191 A2	February 17, 2000	E	033	C12N015/86
AU 9951822 A	February 28, 2000		000	C12N015/86
BR 9912653 A	May 2, 2001		000	C12N015/86
EP 1100942 A2	May 23, 2001	E	000	C12N015/86
GB 2361921 A	November 7, 2001		000	C12N015/869
KR 2001072162 A	July 31, 2001		000	C12N007/01
US 20020099006 A1	July 25, 2002		000	A61K048/00

INT-CL (IPC): A01 N 63/00; A61 K 38/16; A61 K 39/12; A61 K 39/245; A61 K 39/255; A61 K 39/265; A61 K 39/27; A61 K 48/00 ; A61 P 31/00; A61 P 35/00; C07 H 21/04; C12 N 5/10; C12 N 7/01; C12 N 15/10; C12 N 15/86; C12 N 15/869; C12 Q 1/70

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

☐ 9. Document ID: WO 9960145 A1 JP 2002515256 W AU 9939466 A EP 1080215 A1 GB 2359083 A BR 9910594 A CN 1310765 A KR 2001071292 A

L3: Entry 9 of 13 File: DWPI Nov 25, 1999

DERWENT-ACC-NO: 2000-086600
DERWENT-WEEK: 200238

TITLE: Mutant herpes simplex virus useful for the treatment of disease associated with gene function

INVENTOR: COFFIN, R S; LATCHMAN, D S

PRIORITY-DATA: 1998GB-0010904 (May 20, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9960145 A1	November 25, 1999	E	031	C12N015/86
JP 2002515256 W	May 28, 2002		033	C12N015/09
AU 9939466 A	December 6, 1999		000	C12N015/86
EP 1080215 A1	March 7, 2001	E	000	C12N015/86
GB 2359083 A	August 15, 2001		000	C12N015/86
BR 9910594 A	October 30, 2001		000	C12N015/86
CN 1310765 A	August 29, 2001		000	C12N015/86
KR 2001071292 A	July 28, 2001		000	C12N007/01

INT-CL (IPC): A61 K 35/76; A61 K 48/00; A61 P 25/00; C12 N 7/00; C12 N 7/01; C12 N 15/09; C12 N 15/86; G01 N 33/50

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

☐ 10. Document ID: WO 9953043 A2 JP 2002511254 W AU 9934339 A EP 1070123 A2

L3: Entry 10 of 13 File: DWPI Oct 21, 1999

DERWENT-ACC-NO: 1999-633828
DERWENT-WEEK: 200242

TITLE: New polynucleotides used to identify drugs which prevent reactivation of latent Herpes simplex virus (HSV), treating HSV infections

INVENTOR: COFFIN, R S; LATCHMAN, D S ; THOMAS, S K

PRIORITY-DATA: 1998GB-0007865 (April 9, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9953043 A2	October 21, 1999	E	022	C12N015/00
JP 2002511254 W	April 16, 2002		052	C12N015/09
AU 9934339 A	November 1, 1999		000	
EP 1070123 A2	January 24, 2001	E	000	C12N015/00

INT-CL (IPC): A61 K 45/00; A61 P 31/22; C07 K 14/035; C12 N 1/19; C12 N 1/21; C12 N 5/10; C12 N 7/00; C12 N 15/00; C12 N 15/09; G01 N 33/15; G01 N 33/50

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Image

Generate Collection | Print

Terms	Documents
Coffin R S.in.	13

Display Format: CIT Change Format

Previous Page Next Page

1: Gene Ther 2001 Dec;8(24):1840-6

Related Articles, Links

nature publishing group

Prevention of restenosis by a herpes simplex virus mutant capable of controlled long-term expression in vascular tissue in vivo.

Skelly CL, Curi MA, Meyerson SL, Woo DH, Hari D, Vosicky JE, Advani SJ, Mauceri HJ, Glagov S, Roizman B, Weichselbaum RR, Schwartz LB.

Section of Vascular Surgery, Department of Surgery, University of Chicago, Chicago, IL 60637, USA.

Neointimal hyperplasia resulting from vascular smooth muscle cell (SMC) proliferation and luminal migration is the major cause of autologous vein graft failure following vascular coronary or peripheral bypass surgery. Strategies to attenuate SMC proliferation by the delivery of oligonucleotides or genes controlling cell division rely on the use of high concentrations of vectors, and require pre-emptive disruption of the endothelial cell layer. We report a genetically engineered herpes simplex virus (HSV-1) mutant that, in an in vivo rabbit model system, infects all vascular layers without prior injury to the endothelium; expresses a reporter gene driven by a viral promoter with high efficiency for at least 4 weeks; exhibits no systemic toxicity; can be eliminated at will by administration of the antiviral drug acyclovir; and significantly reduces SMC proliferation and restenosis in vein grafts in immunocompetent hosts.

PMID: 11821937 [PubMed - indexed for MEDLINE]

1: Transplantation 1995 Mar 27;59(6):809-16

Multiple vectors effectively achieve gene transfer in a murine cardiac transplantation model. Immunosuppression with TGF-beta 1 or vIL-10.

Qin L, Chavin KD, Ding Y, Favaro JP, Woodward JE, Lin J, Tahara H, Robbins P, Shaked A, Ho DY, et al.

Department of Microbiology, Medical University of South Carolina, Charleston 29425, USA.

The application of gene transfer techniques to organ transplantation offers the potential for modulation of immunity directly within an allograft without systemic side effects. Expression vectors and promoter elements are important determinants of gene transfer and expression. In this study, various vectors (naked plasmid DNA, retroviral vector, herpes simplex viral vector, and adenoviral vector) with various promoters (RSV-LTR, SV40, MuLV-LTR, HCMVie1) were directly compared to demonstrate the successful gene transfer and expression of beta-galactosidase in murine myoblasts in vitro and within murine heterotopic, nonvascularized cardiac isografts or allografts in vivo. Expression of transferred genes was not toxic to cells and strength of expression varied according to the type of vector. Plasmid DNA was expressed in myocytes, retroviral vector was expressed in the graft infiltrating cells, and herpes simplex and adenoviral vectors were expressed in both myocytes and graft-infiltrating cells. Preliminary studies evaluated the ability of these vectors to deliver immunologically important signals. Allografts injected with pSVTGF-beta 1, a plasmid-encoding transforming growth factor beta 1 (TGF-beta 1) under the control of the SV40 promoter, showed significant prolongation of graft survival of 26.3 +/- 2.5 days compared with 12.6 +/- 1.1 days for untreated allografts, and 12.5 +/- 1.5 days for the allografts injected with control plasmid ($P < 0.05$). Allografts injected with MFG-vIL-10, a retroviral vector encoding viral interleukin-10 under the control of the MuLV-LTR, showed prolongation of graft survival of 36.7 +/- 1.3 days versus 12.6 +/- 1.1 days for the untreated allograft, and 13.5 +/- 2.0 days for the allografts injected with control retroviral vector ($P < 0.001$). Both vectors were transcriptionally active in vivo and did not appear to have toxic effects. Gene therapy for

Efficient gene transfer into myocardium by direct injection of adenovirus vectors.

Guzman RJ, Lemarchand P, Crystal RG, Epstein SE, Finkel T.

Cardiology Branch, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Md. 20892.

Previous studies have established that gene transfer into myocardial cells in vivo is detectable after direct injection of plasmid DNA. Recently, adenovirus vectors have been shown to provide an efficient method for gene transfer into a wide range of tissues. Therefore, this study sought to assess the efficiency and stability of adenovirus-mediated gene transfer into myocardium and to compare this method with that using plasmid-based gene transfer techniques. Adult rats underwent myocardial injection via a subdiaphragmatic approach. Gene transfer efficiency was compared using direct injection of an adenovirus vector encoding for the marker gene beta-galactosidase (beta-gal), a control adenovirus vector encoding for the cystic fibrosis transmembrane conductance regulator gene, a plasmid encoding for beta-gal, or a control plasmid. Hearts infected with an adenovirus vector containing the beta-gal gene showed significantly increased beta-gal enzymatic activity compared with hearts injected with beta-gal plasmid. Histological examination revealed that cardiac myocytes were the target of adenovirus-mediated gene transfer. A time course of gene expression showed that beta-gal enzymatic activity peaked during the first week following injection. Adenovirus vectors provide an efficient but transient method for in vivo gene expression in myocardium.

PMID: 8222091 [PubMed - indexed for MEDLINE]

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PubMed Services

Related Resources

1: Skelly CL, Curi MA, Meyerson SL, Woo DH, Hari D, Vosicky JE, Advani SJ, Mauceri HJ, Glagov S, Roizman B, Weichselbaum RR, Schwartz LB. Related Articles, Links

Prevention of restenosis by a herpes simplex virus mutant capable of controlled long-term expression in vascular tissue in vivo.
Gene Ther. 2001 Dec;8(24):1840-6.
PMID: 11821937 [PubMed - indexed for MEDLINE]

2: Reis E, Martinet O, Mosimann F. Related Articles, Links

[Treatment of intimal hyperplasia by gene therapy: an update]
J Mal Vasc. 1999 Dec;24(5):349-55. Review. French.
PMID: 10642646 [PubMed - indexed for MEDLINE]

3: Davies MG, Hagen PO. Related Articles, Links

Pathophysiology of vein graft failure: a review.
Eur J Vasc Endovasc Surg. 1995 Jan;9(1):7-18. Review.
PMID: 7664016 [PubMed - indexed for MEDLINE]

4: Allen KE, Varty K, Jones L, Sayers RD, Bell PR, London NJ. Related Articles, Links

Human venous endothelium can promote intimal hyperplasia in a paracrine manner.
J Vasc Surg. 1994 Apr;19(4):577-84.
PMID: 8164272 [PubMed - indexed for MEDLINE]

5: Davies MG, Barber L, Dalen H, Svendsen E, Hagen PO. Related Articles, Links

Control of the structural and functional consequences of vein graft intimal hyperplasia with a 21-aminosteroid--U74389G.
Eur J Vasc Surg. 1994 Jul;8(4):448-56.
PMID: 8088396 [PubMed - indexed for MEDLINE]

6: George SJ, Angelini GD, Capogrossi MC, Baker AH. Related Articles, Links

Wild-type p53 gene transfer inhibits neointima formation in human saphenous vein by modulation of smooth muscle cell migration and induction of apoptosis.
Gene Ther. 2001 May;8(9):668-76.
PMID: 11406761 [PubMed - indexed for MEDLINE]

☐ **7:** Bryan AJ, Angelini GD. Related Articles, Links

The biology of saphenous vein graft occlusion: etiology and strategies for prevention.
Curr Opin Cardiol. 1994 Nov;9(6):641-9. Review.
PMID: 7819622 [PubMed - indexed for MEDLINE]

☐ **8:** George SJ, Baker AH, Angelini GD, Newby AC. Related Articles, Links

Gene transfer of tissue inhibitor of metalloproteinase-2 inhibits metalloproteinase activity and neointima formation in human saphenous veins.
Gene Ther. 1998 Nov;5(11):1552-60.
PMID: 9930309 [PubMed - indexed for MEDLINE]

☐ **9:** Porter KE, Varty K, Jones L, Bell PR, London NJ. Related Articles, Links

Human saphenous vein organ culture: a useful model of intimal hyperplasia?
Eur J Vasc Endovasc Surg. 1996 Jan;11(1):48-58.
PMID: 8564487 [PubMed - indexed for MEDLINE]

☐ **10:** Chikada M, Jones M. Related Articles, Links

Study of gene delivery in a rabbit vein graft model. Improvement of the efficiency of gene transfer into vein grafts.
Jpn J Thorac Cardiovasc Surg. 1999 May;47(5):204-9.
PMID: 10402767 [PubMed - indexed for MEDLINE]

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Ann Thorac Surg. 1993 Jun;55(6):1522-8.
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Biorheology. 2002;39(3-4):491-9. Review.
PMID: 12122271 [PubMed - indexed for MEDLINE]

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Vein quality influences neointimal hyperplasia in an organ culture model of human saphenous vein.
Eur J Vasc Endovasc Surg. 1997 Jun;13(6):557-62.
PMID: 9236708 [PubMed - indexed for MEDLINE]

☐ **15:** Yasumoto H, Kim S, Zhan Y, Miyazaki H, Hoshiga M, Kaneda Y, Morishita R, Iwao H. Related Articles, Links

Dominant negative c-jun gene transfer inhibits vascular smooth muscle cell proliferation and neointimal hyperplasia in rats.
Gene Ther. 2001 Nov;8(22):1682-9.
PMID: 11892835 [PubMed - indexed for MEDLINE]

☐ **16:** Ohno N, Itoh H, Ikeda T, Ueyama K, Yamahara K, Doi K, Yamashita J, Inoue M, Masatsugu K, Sawada N, Fukunaga Y, Sakaguchi S, Sone M, Yurugi T, Kook H, Komeda M, Nakao K. Related Articles, Links

Accelerated reendothelialization with suppressed thrombogenic property and neointimal hyperplasia of rabbit jugular vein grafts by adenovirus-mediated gene transfer of C-type natriuretic peptide.
Circulation. 2002 Apr 9;105(14):1623-6.
PMID: 11940536 [PubMed - indexed for MEDLINE]

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Genetic engineering of vein grafts resistant to atherosclerosis.
Proc Natl Acad Sci U S A. 1995 May 9;92(10):4502-6.
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Effect of resveratrol on intimal hyperplasia after endothelial denudation in an experimental rabbit model.
Life Sci. 2000 Dec 1;68(2):153-63.
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Angiology. 1995 Feb;46(2):91-7.
PMID: 7702205 [PubMed - indexed for MEDLINE]

☐ **20:** Wilson YG, Davies AH, Southgate K, Currie IC, Knight D, Patton D, Baird RN, Lamont PM, Angelini GD. Related Articles, Links

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J Endovasc Surg. 1996 Nov;3(4):436-44.
PMID: 8959504 [PubMed - indexed for MEDLINE]

Display Summary Show: 20 Sort Send to Text
Items 1-20 of 341 Page 1 of 18 Next

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L3: Entry 13 of 13 File: DWPI Feb 5, 1998

DERWENT-ACC-NO: 1998-130712
DERWENT-WEEK: 200137

TITLE: Herpes simplex virus lacking functional ICP34.5 and ICP27 genes - useful for, e.g. treating injuries to central nervous system such as Parkinson's disease and for gene therapy in mammals

INVENTOR: BROWN, S M; COFFIN, R S ; LATCHMAN, S D ; MACLEAN, A R ; LATCHMAN, D S

PRIORITY-DATA: 1996GB-0015794 (July 26, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9804726 A1	February 5, 1998	E	030	C12N015/86
US 6248320 B1	June 19, 2001		000	A01N063/00
AU 9737007 A	February 20, 1998		000	C12N015/86
EP 920523 A1	June 9, 1999	E	000	C12N015/86
AU 726645 B	November 16, 2000		000	C12N015/86
JP 2000516809 W	December 19, 2000		033	C12N015/00
NZ 333901 A	May 25, 2001		000	C12N007/01

INT-CL (IPC): A01 N 63/00; A61 K 35/30; A61 K 35/34; A61 K 48/00; A61 P 25/00; A61 P 25/16; A61 P 35/00; C07 K 14/035; C12 N 1/21; C12 N 7/00; C12 N 7/01; C12 N 7/04; C12 N 15/00; C12 N 15/86; C12 N 1/21; C12 R 1:01

ABSTRACTED-PUB-NO: US 6248320B
BASIC-ABSTRACT:

A herpes simplex virus (HSV) lacking functional genes ICP34.5 and ICP27, is new.

USE - The HSV strains carrying inactivating mutations in both ICP34.5 and ICP27 genes can be used in the preparation of therapeutic compositions for treating diseases of, or injuries to, the nervous system (claimed), e.g. Parkinson's disease, spinal injury or strokes, or diseases of the eye, heart or skeletal muscles, or malignancies. the strains can be used for gene therapy in humans and animals (claimed). They can also be used for studying the function of genes in mammalian cells (claimed), e.g. identifying genes complementing cellular dysfunctions or studying the effect of expressing mutant genes in wild-type or mutant mammalian cells. The strains may be used in particular for the functional study of genes implicated in disease, e.g. to induce Creutzfeldt-Jakob and other prion-type diseases in the central nervous system of rodents. Other disease models may include those for Alzheimer's disease, motor neuron disease or Parkinson's disease.

ADVANTAGE - The HSV strains carrying both inactivating mutations exhibit greatly improved levels of expression of heterologous genes compared to virus strains carrying mutations in ICP34.5 alone. These doubly-mutated strains are also safer than strains carrying mutations in ICP34.5 alone.

ABSTRACTED-PUB-NO:

WO 9804726A
EQUIVALENT-ABSTRACTS:

A herpes simplex virus (HSV) lacking functional genes ICP34.5 and ICP27, is new.

USE - The HSV strains carrying inactivating mutations in both ICP34.5 and ICP27 genes can be used in the preparation of therapeutic compositions for treating diseases of, or injuries to, the nervous system (claimed), e.g. Parkinson's disease, spinal injury or strokes, or diseases of the eye, heart or skeletal muscles, or malignancies. the strains can be used for gene therapy in humans and animals (claimed). They can also be used for studying the function of genes in mammalian cells (claimed), e.g. identifying genes complementing cellular dysfunctions or studying the effect of expressing mutant genes in wild-type or mutant mammalian cells. The strains may be used in particular for the functional study of genes implicated in disease, e.g. to induce Creutzfeldt-Jakob and other prion-type diseases in the central nervous system of rodents. Other disease models may include those for Alzheimer's disease, motor neuron disease or Parkinson's disease.

ADVANTAGE - The HSV strains carrying both inactivating mutations exhibit greatly improved levels of expression of heterologous genes compared to virus strains carrying mutations in ICP34.5 alone. These doubly-mutated strains are also safer than strains carrying mutations in ICP34.5 alone.

ABSTRACTED-PUB-NO: US 6248320B
EQUIVALENT-ABSTRACTS: A herpes simplex virus (HSV) lacking functional genes ICP34.5 and ICP27, is new. USE - The HSV strains carrying inactivating mutations in both ICP34.5 and ICP27 genes can be used in the preparation of therapeutic compositions for treating diseases of, or injuries to, the nervous system (claimed), e.g. Parkinson's disease, spinal injury or strokes, or diseases of the eye, heart or skeletal muscles, or malignancies. the strains can be used for gene therapy in humans and animals (claimed). They can also be used for studying the function of genes in mammalian cells (claimed), e.g. identifying genes complementing cellular dysfunctions or studying the effect of expressing mutant genes in wild-type or mutant mammalian cells. The strains may be used in particular for the functional study of genes implicated in disease, e.g. to induce Creutzfeldt-Jakob and other prion-type diseases in the central nervous system of rodents. Other disease models may include those for Alzheimer's disease, motor neuron disease or Parkinson's disease. ADVANTAGE - The HSV strains carrying both inactivating mutations exhibit greatly improved levels of expression of heterologous genes compared to virus strains carrying mutations in ICP34.5 alone. These doubly-mutated strains are also safer than strains carrying mutations in ICP34.5 alone. WO 9804726A

CHOSEN-DRAWING: Dwg.0/0

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L3: Entry 12 of 13 File: DWPI Jul 16, 1998

DERWENT-ACC-NO: 1998-399151
DERWENT-WEEK: 200252

TITLE: Vector comprising herpes simplex virus latency-associated transcript P2 region - useful for treating e.g. Parkinson's disease, spinal injury, stroke, etc

INVENTOR: COFFIN, R S; LATCHMAN, D S

PRIORITY-DATA: 1997GB-0000411 (January 10, 1997)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9830707 A2	July 16, 1998	E	026	C12N015/86
AU 749064 B	June 20, 2002		000	C12N015/86
AU 9855669 A	August 3, 1998		000	C12N015/86
BR 9806866 A	April 18, 2000		000	C12N015/86
CN 1250480 A	April 12, 2000		000	C12N015/86
EP 1021553 A2	July 26, 2000	E	000	C12N015/86
MX 9906454 A1	April 1, 2000		000	C12N015/86
KR 2000070037 A	November 25, 2000		000	C12N015/86
JP 2001508294 W	June 26, 2001		035	C12N015/09

INT-CL (IPC): A61 K 35/76; A61 K 39/00; A61 K 39/245; A61 K 48/00; A61 P 9/00; A61 P 25/00; A61 P 25/16; A61 P 27/02; A61 P 31/22; C12 N 7/00; C12 N 7/01; C12 N 15/09; C12 N 15/85; C12 N 15/86; C12 Q 1/68; C12 N 15/09; C12 R 1:92

ABSTRACTED-PUB-NO: WO 9830707A
BASIC-ABSTRACT:

A novel expression cassette comprises a herpes simplex virus (HSV) latency-associated transcript (LAT) P2 region, a promoter and a heterologous gene operably linked in that order. Also claimed are: (1) a nucleic acid vector comprising an expression cassette as above; and (2) a viral strain comprising an expression cassette as above.

USE - The cassette or viral strain containing it can be used in the treatment of a disorder or injury to e.g. the nervous system, especially Parkinson's disease, spinal injury or stroke, diseases of the eye, heart or skeletal muscle or a malignancy (claimed). The expression cassette can also be used to study the function of a heterologous protein is eukaryotic cells (claimed).

ABSTRACTED-PUB-NO: WO 9830707A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/0

1: Circulation 1997 Jul 15;96(2):408-11

Reduction of restenosis after angioplasty in an atheromatous rabbit model by suicide gene therapy.

Steg PG, Tahlil O, Aubailly N, Caillaud JM, Dedieu JF, Berthelot K, Le Roux A, Feldman L, Perricaudet M, Denefle P, Branellec D.

INSERM U-460, Faculte Xavier Bichat, Paris, France.

BACKGROUND: Gene delivery of the thymidine kinase (tk) gene combined with ganciclovir (GCV) limits intimal hyperplasia after abrasion of normal arteries. However, the low efficiency of adenoviral-mediated gene transfer to atherosclerotic arteries has raised concerns about the applicability of this strategy to the prevention of restenosis. METHODS AND RESULTS: A replication-defective adenoviral vector expressing tk (Ad-RSVtk) demonstrated selective toxicity toward GCV-treated arterial smooth muscle cells, with oligonucleolytic cleavage suggesting apoptosis. In vivo, after demonstration of tk expression after Ad-RSVtk delivery, the combination of Ad-RSVtk followed by GCV was tested in a rabbit model of angioplasty of atheromatous iliac arteries. Angioplasty (8 atm, 20 minutes) was performed by use of a hydrogel balloon coated with Ad-RSVtk (4x10(9) plaque forming units). GCV was infused (25 mg.kg(-1) I.V. BID) from days 2 through 7 after angioplasty in 8 of 12 rabbits. Four weeks later, morphometric analysis demonstrated a reduced intima-to-media ratio in the group receiving combination therapy compared with Ad-RSVtk alone (3.0+/-1.2 versus 5.2+/-0.5, P<.018). GCV per se had no effect on intimal hyperplasia after arterial injury. CONCLUSIONS: In vitro, Ad-RSVtk demonstrates selective toxicity toward GCV-treated arterial smooth muscle cells involving apoptosis. In vivo, GCV conditions reduction of neointimal formation after percutaneous delivery of Ad-RSVtk during angioplasty of atheromatous arteries.

PMID: 9244204 [PubMed - indexed for MEDLINE]

WEST

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L6: Entry 1 of 3 File: DWPI Oct 24, 2002

DERWENT-ACC-NO: 2002-537524
DERWENT-WEEK: 200273

TITLE: Expressing heterologous nucleic acid sequence in a vascular cell for treating cardiovascular diseases, involves administering to the cell a genetically engineered herpes simplex viral vector comprising the sequence

INVENTOR: ROIZMAN, B; SCHWARTZ, L B ; WEICHSELBAUM, R R

PRIORITY-DATA: 2000US-253680P (November 28, 2000), 2001US-0995475 (November 28, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20020155432 A1	October 24, 2002		000	C12Q001/70
WO 200245431 A2	June 6, 2002	E	114	C12N000/00
AU 200217885 A	June 11, 2002		000	H04N007/173

INT-CL (IPC): A61 K 39/12; A61 K 39/245; A61 K 39/255; A61 K 39/265; A61 K 39/27; C12 N 0/00; C12 N 15/00; C12 N 15/09; C12 N 15/63; C12 N 15/70; C12 N 15/74; C12 P 21/06; C12 Q 1/70; H04 N 7/173

ABSTRACTED-PUB-NO: WO 200245431A
BASIC-ABSTRACT:

NOVELTY - Expressing (M) a heterologous nucleic acid sequence in a vascular cell, or inducing normal physiology in a functionally abnormal vascular cell, comprising administering to the cell a recombinant replicating herpes simplex viral (HSV) vector having a heterologous nucleic acid, where HSV is debilitated for growth in the central nervous system.

ACTIVITY - Cardiant; Antiarrhythmic; Vasotropic.

To demonstrate in vivo gene transfer in proliferating vascular tissue, the external jugular vein of male New Zealand white rabbits was exposed to vehicle, HSVlacZ (R849), or adeno-associated virus (AAV)lacZ. Male New Zealand white rabbits were anesthetized, the external jugular vein was exposed and two branches cannulated with 24-gauge catheters. One cannula was used for irrigation and infection, and the other for intralumenal pressure monitoring. The main channel was infected with either vehicle, AAVlacZ 4 multiply 1011 plaque forming units (pfu)/ml, or HSVlacZ (R849) 4 multiply 108 pfu/ml for 10 minutes at 100 mmHg. Following infection, the vein was irrigated with saline, excised and bivalved. The ipsilateral common carotid artery (CCA) was exposed through the same incision and the animal systemically anticoagulated with heparin (200 U/kg) intravenously. The CCA was doubly clamped and a 1.5-cm longitudinal arteriotomy made proximal to the cranial thyroid branch. The arteriotomy was reconstructed with external jugular vein patch angioplasty using running 8-0 polypropylene suture. Ultrasonic transit-time flow through the graft was measured. There was no significant difference in mean blood flow in vehicle-treated vs. Viral-infected patches at time of implantation. The incision was closed and the animal was allowed to recover. After four weeks, the vein patches were harvested and assessed for patency and beta -galactosidase expression using X gal. Intraarterial pressure and blood flow through the patch were again measured and recorded. All vein patches that had been exposed to HSVlacZ showed significant beta -galactosidase expression in all layers of the vein wall at 4 weeks after exposure, especially within the smooth muscle cells comprising the neointima (48

plus or minus 2 % infection efficiency). In contrast, patches infected with AAVlacZ showed inconsistent transgene expression, mostly confined to the adventitia. Expression was not evident in the vehicle-exposed patches or in any harvested external jugular veins or CCAs contralateral to an HSVlacZ (R849)-infected vein patch or an AAV-infected vein patch.

MECHANISM OF ACTION - Gene therapy.

USE - (M) is useful for expressing a heterologous nucleic acid sequence encoding a polypeptide such as antiproliferative polypeptide, vasodilatory polypeptide, and an angiogenic polypeptide, an antisense oligonucleotide or antisense polynucleotide complementary to the polypeptide in a vascular cell, such as endothelial cell, smooth muscle cell or adventitial cell. (M) is also useful for inducing normal physiology in a functionally abnormal vascular cell. (M) is useful for treating or preventing a cardiovascular disease or condition such as chronic heart failure, hypertensive cardiovascular disease, ischemic heart disease, arrhythmia, congenital heart disease, valvular heart disease or stenotic defect, cardiomyopathy, aneurysm, chronic venous insufficiency, peripheral arterial disease or restenosis, in a vascular cell. The heterologous nucleic acid sequence is expressed in vascular tissue for a duration of more than 7, 14, 21, 28, 35 or 70 days. The heterologous nucleic acid sequence encodes a screenable or selectable marker, an antithrombotic nucleic acid, angiogenesis regulating nucleic acid, immunomodulator, inducer of cellular proliferation, inhibitor of cellular proliferation or a regulator or programmed cell death. The method further comprises administering at least one pharmacological agent such as antihyperlipoproteinemic agent, antiarteriosclerotic agent, antithrombotic/fibrinolytic agent, blood coagulant, antiarrhythmic agent, antihypertensive agent, vasopressor, treatment agent for congestive heart failure, antianginal agent, anti-infection agent, to the vascular cell. (All claimed).

ADVANTAGE - (M) provides therapeutic benefit both in vascular and cardiovascular tissue.

ABSTRACTED-PUB-NO: WO 200245431A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/0

WEST Search History

DATE: Friday, February 14, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=DWPI; PLUR=YES; OP=ADJ			
L10	herpes virus and acyclovir and gene therapy	2	L10
L9	herpesvirus and acyclovir and gene therapy	0	L9
L8	herpesvirus and vascular and acyclovir and gene therapy	0	L8
L7	herpesvirus and vascular and ganciclovir and gene therapy	0	L7
L6	Schwartz L B.in.	3	L6
DB=USPT; PLUR=YES; OP=ADJ			
L5	SMC and l1	5	L5
L4	L3 and L1	0	L4
L3	angiogen?	9	L3
L2	herpesvirus and vascular and ganciclovir.clm.	7	L2
L1	herpesvirus and vascular and ganciclovir	69	L1

END OF SEARCH HISTORY

1: New Biol 1992 Mar;4(3):238-46

Direct gene transfer to the liver with herpes simplex virus type 1 vectors: transient production of physiologically relevant levels of circulating factor IX.

Miyanohara A, Johnson PA, Elam RL, Dai Y, Witztum JL, Verma IM, Friedmann T.

Department of Pediatrics, University of California, San Diego, La Jolla 92093-0634.

We have used gene transfer vectors derived from a replication-defective mutant of herpes simplex virus type 1 (HSV-1) expressing the hepatitis B virus surface antigen (HBsAg), Escherichia coli beta-galactosidase (beta-gal), or canine factor IX (cFIX) from the immediate early promoter of human cytomegalovirus (hCMV) to infect mouse liver by direct injection or through the portal vein. By either route, high levels of transgene expression were demonstrated by the detection of immunoreactive HBsAg or cFIX in the circulation and by histochemical detection of beta-gal activity in situ. The results were striking in that the serum level of cFIX reached 10% of the normal murine levels. Although the level of transgene expression from the hCMV promoter was transient, a significant number of persistent vectors could be rescued from the livers of recipient mice up to 2 months after inoculation. Replacement of the hCMV promoter with the HSV-1 latency-associated transcript (LAT) promoter resulted in reduced but prolonged expression of both HBsAg and cFIX. The very high level of factor IX expression suggests that clinically useful gene transfer may eventually be feasible through direct vector delivery to the liver.

PMID: 1316145 [PubMed - indexed for MEDLINE]

WEST Search History

DATE: Friday, February 14, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=ADJ			
L12	L6 and intravenous	1	L12
L11	L6 and heptic	0	L11
L10	L6 and aortic	0	L10
L9	L6 and aorta	0	L9
L8	L6 and vein	0	L8
L7	L6 and vascular	0	L7
L6	6509020.pn. and acyclovir	1	L6
L5	6139834.pn. and acyclovir	1	L5
L4	6139834.pn. and ganciclovir	1	L4
L3	6248320.pn. and ganciclovir	0	L3
L2	6248320.pn. and acyclovir	0	L2
L1	6248320.pn. and acylovir	0	L1

END OF SEARCH HISTORY

L5 ANSWER 4 OF 7 MEDLINE
AN 1998426118 MEDLINE
DN 98426118 PubMed ID: 9751684
TI Graft permeabilization facilitates **gene therapy** of transplant arteriosclerosis in a rabbit model.
AU Rekhter M D; Shah N; Simari R D; Work C; Kim J S; Nabel G J; Nabel E G; Gordon D
CS Department of Pathology, University of Michigan, Ann Arbor, MI 48105, USA.
NC HL-43757 (NHLBI)
SO CIRCULATION, (1998 Sep 29) 98 (13) 1335-41.
Journal code: 0147763. ISSN: 0009-7322.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199810
ED Entered STN: 19981029
Last Updated on STN: 19981029
Entered Medline: 19981022
AB BACKGROUND:Smooth muscle cell (SMC) replication plays a central role in the pathogenesis of transplant arteriosclerosis. One strategy to eliminate dividing cells is to express a **herpesvirus** thymidine kinase (tk) gene that phosphorylates the nucleoside analogue ganciclovir into a toxic form leading to cell killing. However, medial SMCs are resistant to gene transfer unless the artery undergoes deendothelialization. We hypothesized that manipulations that increase the "porosity" of the artery can make SMCs prone to gene transfer without denudation. METHODS AND RESULTS:In organ culture of rabbit aorta, longitudinal stretch and supraphysiological pressure applied for 3 hours during incubation with adenoviral vector facilitated gene transfer into medial SMCs without denudation. Of the SMCs, 10.2+/-3.8% expressed a reporter gene of human placental alkaline phosphatase (hpAP), whereas SMCs in control arteries did not express hpAP. To evaluate the feasibility of transgene expression in arterial grafts, we performed such permeabilization-assisted reporter gene transfer into aortas of donor Dutch Belted rabbits and transplanted them into carotid arteries of recipient New Zealand White rabbits. Unstretched transfected grafts were used as a control. SMCs expressed hpAP (7. 3+/-2.4% of cells in 2 days and 4.2+/-1.9% in 2 weeks) in stretched grafts only. In the next series of experiments, we transfected stretched grafts with ADV-tk and combined transplantation with systemic administration of ganciclovir. Stretched ADV-hpAP grafts were used as a control. In 2 weeks, the formation of intimal thickening in tk-expressing grafts was significantly reduced (P<0. 01) because of a decrease in proliferating SMCs. CONCLUSIONS:Manipulations within target tissues can enhance the efficiency of gene transfer into SMCs. Although mechanical permeabilization is clinically problematic, in principle, targeting SMC replication may provide a genetic approach to the treatment of transplant arteriosclerosis.
CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Adenoviridae: GE, genetics
*Aorta: TR, transplantation
Arteriosclerosis: ET, etiology
*Arteriosclerosis: TH, therapy
Cell Division
Ganciclovir: TU, therapeutic use

d l5 1-7

L5 ANSWER 1 OF 7 MEDLINE
AN 2002092052 MEDLINE
DN 21679192 PubMed ID: 11821937
TI Prevention of restenosis by a herpes simplex virus mutant capable of controlled long-term expression in **vascular** tissue in vivo.
AU Skelly C L; Curi M A; Meyerson S L; Woo D H; Hari D; Vosicky J E; Advani S J; Mauceri H J; Glagov S; Roizman B; Weichselbaum R R; Schwartz L B
CS Section of Vascular Surgery, Department of Surgery, University of Chicago, Chicago, IL 60637, USA.
NC 5T32 HL07237 (NHLBI)
RO1 CA71933 (NCI)
SO GENE THERAPY, (2001 Dec) 8 (24) 1840-6.
Journal code: 9421525. ISSN: 0969-7128.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200203
ED Entered STN: 20020201
Last Updated on STN: 20020324
Entered Medline: 20020322

L5 ANSWER 2 OF 7 MEDLINE
AN 2001543311 MEDLINE
DN 21475979 PubMed ID: 11591892
TI Combination **vascular** delivery of herpes simplex oncolytic viruses and amplicon mediated cytokine gene transfer is effective therapy for experimental liver cancer.
AU Zager J S; Delman K A; Malhotra S; Ebright M I; Bennett J J; Kates T; Halterman M; Federoff H; Fong Y
CS Department of Surgery, Hepatobiliary Division, Memorial Sloan-Kettering Cancer Center, New York, New York 10021, USA.
NC RO1 CA 61524 (NCI)
RO1 CA 72632 (NCI)
RO1 CA 75416 (NCI)
SO MOLECULAR MEDICINE, (2001 Aug) 7 (8) 561-8.
Journal code: 9501023. ISSN: 1076-1551.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200202
ED Entered STN: 20011010
Last Updated on STN: 20020215
Entered Medline: 20020214

L5 ANSWER 3 OF 7 MEDLINE
AN 1999321302 MEDLINE
DN 99321302 PubMed ID: 10395379
TI Liposomal encapsulation of ganciclovir enhances the efficacy of herpes simplex virus type 1 thymidine kinase suicide **gene therapy** against hepatic tumors in rats.
AU Engelmann C; Panis Y; Bolard J; Diquet B; Fabre M; Nagy H; Soubrane O; Houssin D; Klatzmann D
CS CHU Pitie-Salpetriere, Paris, France.
SO HUMAN GENE THERAPY, (1999 Jun 10) 10 (9) 1545-51.
Journal code: 9008950. ISSN: 1043-0342.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199908
ED Entered STN: 19990910
Last Updated on STN: 19990910
Entered Medline: 19990826

L5 ANSWER 4 OF 7 MEDLINE
AN 1998426118 MEDLINE
DN 98426118 PubMed ID: 9751684
TI Graft permeabilization facilitates **gene therapy** of transplant arteriosclerosis in a rabbit model.
AU Rekhter M D; Shah N; Simari R D; Work C; Kim J S; Nabel G J; Nabel E G; Gordon D
CS Department of Pathology, University of Michigan, Ann Arbor, MI 48105, USA.
NC HL-43757 (NHLBI)
SO CIRCULATION, (1998 Sep 29) 98 (13) 1335-41.
Journal code: 0147763. ISSN: 0009-7322.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199810
ED Entered STN: 19981029
Last Updated on STN: 19981029
Entered Medline: 19981022

L5 ANSWER 5 OF 7 MEDLINE
AN 97034290 MEDLINE
DN 97034290 PubMed ID: 8879946
TI **Gene therapy** for ischemic heart disease.
AU Malosky S; Kolansky D M
CS Cardiovascular Division, Hospital of the University of Pennsylvania, Philadelphia 19104, USA.
SO CURRENT OPINION IN CARDIOLOGY, (1996 Jul) 11 (4) 361-8. Ref: 63
Journal code: 8608087. ISSN: 0268-4705.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199701
ED Entered STN: 19970219
Last Updated on STN: 19970219
Entered Medline: 19970117

L5 ANSWER 6 OF 7 MEDLINE
AN 95136442 MEDLINE
DN 95136442 PubMed ID: 7530606
TI Expression of **vascular** endothelial growth factor from a defective herpes simplex virus type 1 amplicon vector induces angiogenesis in mice.
AU Mesri E A; Federoff H J; Brownlee M
CS Department of Medicine, Albert Einstein College of Medicine, Bronx, NY 10461.
SO CIRCULATION RESEARCH, (1995 Feb) 76 (2) 161-7.
Journal code: 0047103. ISSN: 0009-7330.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199502

ED Entered STN: 19950314
Last Updated on STN: 19960129
Entered Medline: 19950228

L5 ANSWER 7 OF 7 MEDLINE
AN 94323756 MEDLINE
DN 94323756 PubMed ID: 8047883
TI **Gene therapy** for **vascular** smooth muscle cell proliferation after arterial injury.
CM Comment in: Science. 1994 Aug 5;265(5173):738
AU Ohno T; Gordon D; San H; Pompili V J; Imperiale M J; Nabel G J; Nabel E G
CS Howard Hughes Medical Institute, University of Michigan Medical Center, Ann Arbor 48109.
NC AI33355 (NIAID)
HL43507 (NHLBI)
SO SCIENCE, (1994 Aug 5) 265 (5173) 781-4.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199409
ED Entered STN: 19940909
Last Updated on STN: 19940909
Entered Medline: 19940901

> d l21 1-2

L21 ANSWER 1 OF 2 MEDLINE
AN 95136442 MEDLINE
DN 95136442 PubMed ID: 7530606
TI Expression of vascular endothelial growth factor from a defective herpes simplex virus type 1 amplicon vector induces **angiogenesis** in mice.
AU Mesri E A; Federoff H J; Brownlee M
CS Department of Medicine, Albert Einstein College of Medicine, Bronx, NY 10461.
SO CIRCULATION RESEARCH, (1995 Feb) 76 (2) 161-7.
Journal code: 0047103. ISSN: 0009-7330.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199502
ED Entered STN: 19950314
Last Updated on STN: 19960129
Entered Medline: 19950228

L21 ANSWER 2 OF 2 MEDLINE
AN 92089027 MEDLINE
DN 92089027 PubMed ID: 1751452
TI Selective elimination of recombinant genes in vivo with a suicide retroviral vector.
AU Plautz G; Nabel E G; Nabel G J
CS Howard Hughes Medical Institute, University of Michigan Medical Center, Department of Internal Medicine, Ann Arbor 48109-0650.
NC AI 29179 (NIAID)
DK 42706 (NIDDK)
GM-13457 (NIGMS)
SO NEW BIOLOGIST, (1991 Jul) 3 (7) 709-15.
Journal code: 9000976. ISSN: 1043-4674.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199201
ED Entered STN: 19920216
Last Updated on STN: 19920216
Entered Medline: 19920128

=> d l21 1-2 ab

L21 ANSWER 1 OF 2 MEDLINE
AB Vascular endothelial growth factor (VEGF) is a secreted endothelial cell-specific **angiogenic** growth factor. VEGF gene transfer strategies to stimulate focal **angiogenesis** could be used to ameliorate myocardial ischemia. To induce **angiogenesis** in vivo, we have constructed a replication-defective herpes simplex virus type 1 (HSV-1) amplicon vector that places the human VEGF-165 cDNA under the transcriptional control of the HSV immediate-early 4/5 promoter (HSVhvegf). Transduction of NIH 3T3 fibroblasts with HSVhvegf resulted in the secretion of high levels of biologically active VEGF, as assayed by microvascular endothelial mitogenesis. By use of an ex vivo protocol, BLK-CL4 fibroblasts were transduced with HSVhvegf or control HSVlac virus (expressing Escherichia coli beta-galactosidase), resuspended in basement membrane extract (matrigel), and coinjected subcutaneously into syngeneic C57BL/6 mice. One week later, the matrigel plugs with HSVhvegf showed a

strong **angiogenic** response, in contrast to the plugs with HSVlac-transduced fibroblasts. These data indicate that transduction with HSVhvegf virus can induce an **angiogenic** response in vivo and suggest that this is a viable **gene therapy** approach for tissue ischemia.

L21 ANSWER 2 OF 2 MEDLINE

AB The ability to express recombinant genes in vivo offers potential new treatments for human disease if questions of safety and toxicity can be addressed. Complications of gene transfer could include, for example, overexpression of introduced genes for growth or **angiogenic** factors or insertional mutagenesis, both of which could cause uncontrolled cell growth. We report the development of a suicide retroviral vector that provides a method to eliminate cells undergoing rapid growth in vivo. A murine amphotropic retroviral vector was constructed in which the gene for **herpesvirus** thymidine kinase was included to render proliferating cells sensitive to ganciclovir, and the Escherichia coli beta-galactosidase gene served as a reporter. This vector's efficacy was first assessed in vitro, and beta-galactosidase activity was abolished in several cell lines after treatment with ganciclovir. In vivo, a transplantable murine CT26 adenocarcinoma whose cells were transduced with this vector regressed completely after administration of ganciclovir. In contrast, expression in nondividing cells within rabbit arteries transduced by retroviral infection in vivo was unaffected. This suicide vector therefore eliminates transformed cells but allows survival of normal nondividing cells that express its specific recombinant genes in vivo, and may thus improve the safety and efficacy of gene transfer into living organisms.

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(FILE 'HOME' ENTERED AT 13:18:40 ON 14 FEB 2003)

FILE 'MEDLINE' ENTERED AT 13:19:12 ON 14 FEB 2003

L1	20015	S	GENE THERAPY
L2	33811	S	HERPESVIRUS
L3	474	S	L1 AND L2
L4	298872	S	VASCULAR
L5	7	S	L3 AND L4
L6	31	S	ACYCLOVIR AND L4
L7	6	S	L6 AND L2
L8	84	S	ACYCLOVIR AND L1
L9	28	S	L8 AND L2
L10	1	S	L4 AND L9
L11	0	S	HEART AND L9
L12	8534	S	VASCULAR SMOOTH MUSCLE CELL?
L13	2	S	L12 AND L1 AND L2 AND L4
L14	3215	S	SMC
L15	2	S	L14 AND L1 AND L2
L16	1	S	L14 AND L1 AND L2 AND L12
L17	4087	S	GANCICLOVIR
L18	1	S	L14 AND L1 AND L2 AND L17
L19	1	S	ANGIOGENISIS
L20	16815	S	ANGIOGEN?
L21	2	S	L1 AND L2 AND L20